It seems remarkable that the cases that have spontaneously become bacteria-free have hitherto escaped detection.

I believe I have brought forward sufficient evidence to prove that subacute bacterial endocarditis is a disease in which healing can occur from the bacteriological, pathological, and clinical standpoints, although the evidence of complete recovery from the clinical side is still very meagre.

It is a pleasant duty for me to express my gratitude to Drs. Rudisch, Meyer, Brill, and Manges, the visiting physicians to Mount Sinai Hospital, for their kindness during many years in permitting me to make studies on their cases. The clinical data are mainly derived from the cases in the service of Dr. Rudisch and from cases which a number of physicians permitted me to observe in their practice. I wish to add that it would not have been possible to complete the bacteriological and pathological studies as far as they have been carried without the enthusiastic assistance of the various members of the laboratory staff for the last ten years.

GLOMERULAR LESIONS OF SUBACUTE BACTERIAL ENDOCARDITIS.[1]

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In the kidneys of nearly all individuals dying during the course of subacute bacterial endocarditis there exists a pathological lesion which affects one or more loops of a variable proportion of the glomeruli.[2]

The first to draw attention to this lesion and to connect it with infections of the endocardium by the endocarditis coccus (Streptococcus viridans) was Loehlein. In March, 1910, he reported 8 cases of this condition, in all of which loops of some of the glomeruli showed the lesions. Three of these showed organisms in blood cultures taken during life, and in one of these, organisms were cultivated from vegetations post mortem. In one case the lumen of an artery entering an infarcted area was occluded by an embolus which contained numbers of cocci.

Before proceeding to a consideration of the glomerular lesions it is necessary to understand a few of the cultural characteristics of the endocarditis coccus in order to be able to appreciate the mechanism by which the kidney lesions are produced. On the surface of agar, its growth is exceedingly dry and sometimes difficult to remove. When an attempt is made to emulsify this growth in normal salt solution, the bacteria show a very marked tendency to hang together in small firm clumps which usually cannot be broken up even by very vigorous shaking.

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[1] Read by invitation at the meeting of the Association of American Physicians, May 15, 1912.

[2] Baehr, Jour. Exp. Med., xv, 330.

Smears made from pieces of vegetations in these cases when squeezed between slides and stained by Gram's method, usually show the presence of the typical cocci in numbers such as are seen only in smears made from pure cultures. It is quite conceivable, therefore, that large numbers of these clumps are constantly being washed off the vegetations by the blood current, and being just small enough to pass the vasa afferentia in the kidney, become lodged in the capillary loops of the glomeruli.

In the kidneys of individuals dying during the active bacterial stage of the disease there are seen, as a rule, all stages of the glomerular lesions. The number of involved loops of a glomerular tuft varies in the different diseased Malpighian bodies. Furthermore, there is a great variation in the relative number of glomeruli involved in different parts of the kidneys. The uninvolved glomeruli and the uninvolved loops of involved glomeruli show no changes.

The very earliest stage of the process appears to consist of a swelling of the glomerular epithelium in the involved loops. This may be situated at any portion of the glomerular tuft. If the lesion is large, a segment of the glomerulus, or even the entire Malpighian body may be involved by the process. At first the outlines of the swollen epithelial cells in the diseased segment can be made out. Subsequently they are lost and the entire structure is fused into a homogeneous, finely granular material. In this mass some of the nuclei are fading or undergoing karyorrhexis, although a few may still remain fairly well preserved.

If the deep staining, finely granular mass is situated at the periphery of the glomerulus, the epithelium of the visceral layer of Bowman's capsule over the involved part is usually found to be swollen, proliferating, and desquamating into the capsular space. When the mass with its overlying epithelium comes into contact with the parietal layer of Bowman's capsule, the latter then probably also takes a share in the desquamation, and perhaps in the proliferation.

Eventually this epithelium undergoes necrosis, as did the previously swollen epithelium of the involved loops of the glomerulus. It also becomes part of the homogeneous, finely granular mass which now fuses with the parietal layer of Bowman's capsule. At this stage, the mass may become flattened out over the area of fusion with Bowman's capsule in a crescentic fashion.

The homogeneous cellular material is seen to be traversed by a very delicate network. Eventually the entire mass becomes thoroughly organized. At the same time there occurs active growth of the epithelial cells of the adjacent intact portions of the parietal layer of Bowman's capsule. They advance over the inner aspect of the necrotic mass which by this time has usually separated somewhat from the rest of the glomerulus. Eventually the entire lateral surfaces of the mass are covered by this invading epithelium.

The healed stage of the lesion presents the following picture: A hyaline mass having roughly the shape of a truncated pyramid; its base fused with the adjacent interstital tissue; its mesial aspect adherent to the remainder of the glomerulus; its sides clothed by a reflection from the adjacent epithelium of Bowman's capsule.

When the entire glomerulus is involved, the peripheral portion may be seen to be replaced by concentric fibrils of connective tissue, whereas the central portion still consists of a homogeneous material. In the same section, however, examples of even later stages are frequently seen, the glomerulus being entirely replaced by a mass of hyaline fibrous tissue.

Practically every stage of the process described above is to be seen in nearly every microscopic section of the kidney cortex from most of the cases, and this association of the various stages is an important part of the pathological picture.

The typical lesions were found in 23 of the 25 cases due to the endocarditis coccus. In 2 cases, 1 of which was due to the influenza bacillus and 1 due to the gonococcus, the lesions were not found. The percentage of glomeruli involved varied from 2 to 75 per cent. in various cases.

In sections of 5 cases in which portions of the kidneys were fixed in alcohol, Gram-Weigert stains demonstrated bacterial emboli in the capillaries of the glomeruli. In 2 cases they were easily found; in the other 3, they were only discovered after prolonged search.

The changes that occur in the tubules and interstitium and in the end may lead to a picture identical with that of the secondary contracted kidney are discussed in the paper already referred to. In the same paper will be found a description of the atypical lesions which were found in 2 cases and which closely resembled the lesions found in subacute glomerular nephritis.

In 54 cases of acute endocarditis due to the ordinary pathogenic bacteria, none of the glomerular lesions were found in any case. In the cases which Dr. Libman has described as being instances of a healed or bacteria-free stage of subacute bacterial endocarditis; the characteristic lesions were found every time, only the healed stage of the lesion being present. In these cases the lesions were less abundant than in cases in which the endocarditis coccus had been found during life or after death.